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OF THE CHILDREN'S HOSPITAL

WASHINGTON, D. C.

PSYCHOLOGICAL PROBLEMS IN THE MANAGEMENT OF A DIABETIC CHILD.
Belinda C. Straight, M.D., Katherine S. Meyer, M.S.S., Eve Gewirtz, Ph.D., James Hatleberg, M.D., and Earle Silber, M.D. 55

LACTIC DEHYDROGENASE IN THE CEREBROSPINAL FLUID OF CHILDREN: A PRELIMINARY REPORT.
Ruth K. Jacoby, M.D. 62

MUSHROOM POISONING.
Joseph C. Evers, M.D. 66

CLINICAL BRIEF: A CASE OF CAT SCRATCH DISEASE COMPLICATED BY ERYTHEMA NODOSUM.
Lillian K. Ziegler, M.D. 71

THE EDITOR'S COLUMN: OPERATING TIME IS IMPORTANT 74

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Psychological Problems in the Management of a Diabetic Child

Belinda C. Straight, M.D.,* Katherine S. Meyer, M.S.S.,† Eve Gewirtz, Ph.D.,‡ James Hatleberg, M.D.,§ and Earle Silber, M.D.¶

Dr. Silber:

This case illustrates some of our goals in working with children who have a chronic disease such as diabetes. I think that it is important to help the child accept realistically what having a chronic illness means in terms of the changes that are indicated in his life. It is not helpful to pretend that the illness does not make the child different; it is better to help the child accept the fact that there is a difference, and to learn about what can be done to live with the difference.

Dr. Straight:

This 10 year old boy was referred to the Psychiatry Department by the internist who had been treating him since 1954 when he was first discovered to have diabetes. The initial referral was for a behavior problem of frequent temper tantrums and aggressive behavior such as kicking and biting his teachers, mother, grandmother and grandfather. He was also not eating well. He was not taking his insulin regularly and was breaking his diet. These factors made it difficult to keep his diabetes under control.

The patient was a handsome, bright-eyed, stubbly-haired boy. He came willingly with me into the playroom, talking rapidly and relating very easily. He said he had come to Children's Hospital to have his blood sugar taken because he had diabetes. He was mad because the teacher at school said that there was another boy in his class who had diabetes worse than he. "If she's so smart, how does she know?" The first grade teacher hit him on the head with a ruler, he said. "Didn't she realize that a boy with diabetes shouldn't be treated that way?"

He then told long stories of how people mistreated him. He said at one

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point in the interview, "Do you think there will be a cure? Will I outgrow diabetes?" And when I expressed some doubt that he would outgrow it, he insisted he would either outgrow it or there would be a cure.

He said, "I live with my mother. My mother is divorced, but she doesn't want anybody to know it because they will be mad at her if they know it." When asked whom he would like to take on a desert island with him, (a question we ask to get an idea of who is most important to a child), he said, "I'd take an island bird, one who knew his way around and who could talk to me and tell me things." (This suggested a lack of trust in people.) When asked about dreams, he said, "Yes, I have a dream that comes over and over again, one where my doctor tells me that I can eat anything I want." He has another recurrent bad dream that a certain boy is after him. His best friend is so selected because he is bigger than his enemy and can beat up his enemy for him.

A day or so later I was called by the ward nurse who said the patient was having a temper tantrum and had locked himself in the bathroom. When I arrived on the ward, his mother was sitting in the bathroom on a chair pleading with him to come out of a cubicle into which he had locked himself. He began screaming and pounding on the cubicle door, saying, "Go away, I want to be alone!" Before locking himself in the bathroom, he had actually tried to get on the elevator to go home. He is one of the few children, incidentally, who has really tried to leave the hospital. He emerged from the bathroom and ran screaming down the hall to his room. There he bit his mother on the arm. After I stopped him, I accompanied his mother out of the room. When I attempted to re-enter the room, he had barricaded the door with a chair and a bed. However, it was not barricaded too well, and after forcing my way in I found he had locked himself in the small bathroom. He cried that he wanted his bird; he wanted his grandfather; he wanted to go home for an hour; he hated doctors. When I told him that he would have to stay until the doctor said he could go home when he was well enough, he ceased screaming suddenly and came out of the bathroom very passively. There was obvious relief that someone had taken over the situation for him. Incidentally, a blood sugar determined soon after this episode was 25 mg. per 100 ml. However, this type of behavior had been previously noted at other times when his blood sugar was normal.

I felt that this was a boy with a great deal of anxiety about his illness, who was using his illness to control the adults around him, and that in being able to control adults, his own anxiety increased so much that he became both aggressive and destructive. It was clear that he would need psychotherapy in accepting his illness, so that he would not undermine his diabetic controls and endanger his life.

Mrs. Meyer:

This child was referred to us in February 1957. He had been living with his mother only since September 1956. Prior to that time both he and his mother had been living with the maternal grandparents in their Washington home. Apparently both mother and child were treated like children by the grandparents who made all the decisions. It was not until the mother flatly put her foot down when the boy was eight and said that he must walk to school alone, that the grandfather had stopped taking him to school in the car. The grandmother had such great anxiety about any freedom or opportunity for the boy to play with other children that she never allowed any of his friends in the house. The grandparents began looking around for a smaller home in the country subsequent to the grandfather's retirement from his job. The home had actually been bought and the moving day set before the mother was told that they were going to move. So, quite unceremoniously, mother and son had to look for an apartment.

There were no plans about his care before and after school. For example, when he was truant from school, the mother might not know it, and if she did find out about it, might not be very concerned. Until the time of the move, the grandfather would take up all school matters when he took the boy to school. Consequently, the mother was quite overwhelmed when the school said that he had a serious behavior problem and they believed he should be put in a special class. After school, the boy spent some time at his mother's office. He would also spend some after-school time with an adult post-polio patient in the apartment building in which they lived, but there was never any structured time for play with other boys.

Occasionally, the mother would take him and one or two of his friends roller skating, but he did not like to roller skate and it was pretty much a matter of her choice rather than something he wished. His mother is a 33 year old secretary and stock clerk in a small mail order house where she earns about fifty dollars a week. When they first moved into their apartment, she tried to find someone who would take care of him after school, but as soon as the prospective baby-sitter heard that he had diabetes, she changed her mind.

The father has not been living with the family since shortly after the child's birth. The parents were divorced when he was about 2 years of age. The father has not seen his son since he was 6. He contributes irregularly to the boy's support, is reported to be married again and to have another child. There are many fantasies in regard to what and who his father is.

When discharged from the hospital in February he went to live with his grandparents, since his mother felt that she could not cope with him. From his grandparents' home, he went to the Christ Child Convalescent Home in

March. Since May, both mother and child have been seen in our department on a once weekly basis, the mother and child coming at the same time so as to give them a small interval of visiting before and after their individual appointments.

The mother feels that she is really not a mother and does not know how to take care of him. When she told me about the occasion when sugar was first detected in the urine, she said her first reaction was "What have I done to the child?" When he went to live with his grandparents after his hospitalization here, both mother and grandparents were resentful that he had been sent to live with them. (In any dissension which arises in trying to control the boy, the grandparents fall apart.) They report threats they have made to him; they walk out on him and these episodes are followed by tears on the part of the grandparents. During this interval, the grandparents used to repeat stories to him about the horrible things that happen to diabetics who do not obey instructions. Stories about relatives who had been blind, or who had died as a result of eating too much candy were elaborated with examples such as "If you don't mind, you'll get diabetes, or go blind or you'll die."

The mother described the grandparents as great worriers. They could have no life of their own, either with friends, relatives or neighbors. They furthermore had not let the mother have any life when she was growing up. The mother was, incidentally, an adopted child, and at the age of 5 became so unsatisfactory to the grandparents that they tried to return her to the institution from which she had been adopted.

Since the patient's stay at the Christ Child Home, the Home has complained at various times about such things as his fighting with other children in such a way as not to get caught, dawdling excessively, and disregarding requests to tidy his room. He immediately took advantage of every small privilege allowed him because of his diabetes. He spent much time reporting adversely to mother and grandparents about the Home, saying he was not getting enough to eat or was getting too much, or was not getting his insulin on time, and in general, made so many complaints that it threw his relatives into a tailspin. Following an appendectomy in June, the boy again went to live with the grandparents, since the Home felt there then were too many active children there for him to stay.

Since August, the mother has reported that he has not been so anxious nor has she been receiving so many contrived complaints as before. She has now begun to discuss realistic planning for her son. There is a great deal of interest expressed as to where he is going to live. Alternatives which have been mentioned include an institutional school-type of placement, or both mother and child living in a combined adult and child foster home with the

foster home not taking on quite the same responsibility for the boy's care as it would if he were separated from the mother.

Dr. Gewirtz:

Psychological tests were administered to the patient in two parts. He was first seen in his room in the hospital in February and was given a number of projective tests. He was extremely restless and irritated, trying to cooperate but finding it hard to sit still, talking all the time and abruptly, and in general, ridiculing and belittling the test situation as well as himself. When asked to draw a person, he tried to make the characters "funny" with "block heads" and referred to them as his teacher, friends at school or himself.

Of the 10 cards presented during the Rorschach test, he would only respond to six. Of those to which he responded, he saw masks, cannibals and TV programs, all being responses given in a very casual belittling way. None had color as a determinant and some were quite poor in terms of the reality features of the card. When he was given more structured pictures in the Michigan picture test, he said he did not like to make up "silly stories" and described only "one little boy who looks scared," but refused to say why.

Later in June, he was given a Wechsler intelligence test. At that time, he was generally more relaxed but still uneasy, and seemed very concerned about what he thought were failures. He tried to belittle the test, particularly when he felt uneasy about it, but was equally pleased when he had a good response. When praised for his good memory on one problem involving digits, he said, "Oh, don't say that, because then I won't remember any more," and indeed he missed the next item. While he attained a total average IQ of 106, there was a discrepancy between his IQ on the verbal part of the test (116) and the performance part (96), during which he appeared considerably more restless and uneasy.

The general impression derived from these tests is that of a child who feels extremely anxious, insecure and inadequate, and is particularly panicked by any emotion-arousing situations with which he can not cope. Consequently, most of his emotional responses appear to be markedly suppressed. He seems to adopt a distant and ridiculing attitude as a defense against any serious communication. He appears to be particularly anxious when the interaction involves close personal contact between himself and another person. His emotional concerns are likely also to restrict the range of his intellectual functioning.

Dr. Hatleberg:

The previous discussants have presented a picture of the basic personality problems which this child has and how the diabetes has further complicated

his life. A very important occurrence for him began when he started regularly to see the resident assigned to psychiatry. He had been told many things about diabetes and had many questions and fears about it, but had never previously met a significant adult person to whom he could talk about diabetes and not have the adult be afraid. He was particularly worried whether diabetes was contagious, and whether he was different from other people because he had diabetes. In his contact with the resident, he became more relaxed about his illness, but at the same time continued to test the resident, and more recently, me, to determine just what sort of person the doctor is. The doctor is an important person to the child because the doctor who understood diabetes would have to be a person he could trust if he is to be able to overcome his fears. Our playroom situation is set up so that the child had the opportunity to have the attention of the doctor, learn to know him, and to focus on his own problems.

In the playroom interviews with the resident, he could tolerate the interview up to the point where he began to be aggressive. On one occasion, he picked up a dart gun and, whether accidentally or not, shot the gun and hit the resident; that was the end of the playroom for him. He began following a pattern of going around the hospital and especially to the ward where he had previously been hospitalized, and attempting to involve other people in the hospital in his treatment situation rather than to be alone the entire hour with only one person, the resident. When the resident finished his period of training and left the hospital, the boy had a period of mourning because this adult who had been so kind to him had left. He had feelings that he had done something wrong and had been a bad boy. It took him a period of several hours to talk things over and finally realize that his friend had not left him because he had been bad but rather because the resident had other plans to further his career.

The first time I saw him, he was quite anxious and again afraid to go into the playroom. He told me that he was a diabetic and mentioned some of his ideas about diabetes. He said, "You know, I am lucky that I have diabetes, because when you have diabetes and take good care of yourself, you live longer than people who don't have diabetes. My aunt, you know, she had diabetes and she ate some sugar and she died." I think here we see just what his attitude was toward his illness. There is an attempt at denial that the illness is a serious one; he has to say that he is lucky to have it.

This boy has, however, not really focused a lot on the diabetic problem. His problem has been more in the major area of controlling people. Diabetes is, of course, a problem because if he is not in control he is anxious, since he feels his illness can control him.

In the beginning, he continued to roam around the hospital but when we

were in the playroom, he became interested in some of the toys that were there. He brought in some card games and showed me some tricks. From being able to trick me in the card game, we started playing simple card games. He then graduated to chess and bingo, and as he became more involved in the games he was quite anxious at first and I pointed out to him that it was important for him to win. He said, "If you win, I'll shoot you." He also showed some concern that if he would win I would beat him up. He gradually came to realize that they were only games and his winning did not make me angry at him. His aggression is very definitely a part of controlling and winning.

One other place where his aggression is manifest is in his separation from his mother. When I asked him who would determine how long he would be staying at the Christ Child Home, he said he didn't know. He thought probably it would be a medical decision. So we talked this over and gradually he came to realize that his mother is not able to plan and provide for him, and that the reason she visits the psychiatry department is to get help so she can adequately plan for him. Elaborating further, I reassured him that no one is going to take his mother away from him, and that even though he is presently separated from her, his mother will always be available to him. I said that I knew it is difficult for him when he is not with his mother. He replied that it was, and that he thinks about it quite a bit. In fact, he has trouble going to sleep at night when he thinks about being away from her.

In summary, I believe this is a boy with excessive feelings of helplessness when he is not in control of the situation or people, and that the chronic illness, in this case diabetes, complicates the feeling of helplessness.

Dr. Silber:

This boy attempted to deal with his diabetes by denying that the illness was as serious as it actually was. On the other hand, one certainly does not want to flood a child with anxiety about his illness so that it renders him feeling helpless, and gives him the impression that nothing can be done in the way of constructive living with a chronic disease. I think this case also points out the importance of the child's being able to have a sense of trust in the physician; he needs someone who can acquaint him with facts about his illness, and someone who will deal with him realistically about it. Ultimately the child's security about himself can grow out of this kind of realistic adaptation to the illness and its stresses. If he feels he cannot trust adults to provide the kind of care he needs, he feels threatened. If he feels that adults will give in to him when it is not indicated, for example, in terms of making allowances in diet, he again feels threatened. This was the case with this boy at the beginning of his illness. He found it very difficult,

I think, to have any sense of trust in his mother because she felt it was helpful to him for her to make special allowances in his diet which were inappropriate.

Another aspect of this problem mentioned over and over again is the question of control. I think this child has not had sufficient experience with a sense of trust in the ability of adults to plan and care for him. He tends to feel that in any situation in which he is not in control he is threatened. He has not known of adults with whom he could relax. The need to feel in control of the situation, to feel that he must control adults, I think will lessen as he can feel more and more a sense of trust in his relationship with an adult.

There were many situations that also arose in the course of working with this child that illustrated his tendency to deal with aggressive feelings in a very self-destructive way. For example, when angry at someone he would refuse to eat, and in turn would be apt to go into insulin shock as a result, so that his difficulty in dealing with aggression became a very serious problem in terms of the medical management of the diabetes. Another important aspect of his psychotherapy is, therefore, to help him find ways to deal with his aggressive feelings that are not self-destructive, ways that can help him adjust to his illness and adapt to living with other people. He might be encouraged, for example, to use verbalization rather than to refuse to eat as a way of dealing with his hostility.

This boy presents some rather extreme problems, but they highlight some of the every day kinds of problems that I am sure all of you must see in your work with children when you are faced with the task of helping them integrate chronic diseases into their lives.

Lactic Dehydrogenase in the Cerebrospinal Fluid of Children: a Preliminary Report

RUTH K. JAKOBY, M.D.*

The utilization of serum enzymes as indicators of tissue necrosis and ischemia has received great impetus as the result of Karmen's[1] development of the glutamic-aspartic transaminase assay which has been used to detect myocardial infarction[2] and liver damage.[3] In an analogous manner it was argued that cases of central nervous system pathology may allow diffusion

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of enzymes from brain and spinal cord into the CSF.† Using lactic dehydrogenase as an indicator, Jakoby and Jakoby[4] were able to demonstrate high titers of the enzyme in the CSF of patients with cerebrovascular disease and with meningitis. Indeed, this method was shown to be applicable in differentiating cerebrovascular disease from brain tumor, conditions whose symptoms occasionally mimic each other.

Because of the apparent usefulness of the method in adults a survey has been conducted on lactic dehydrogenase in the CSF of young children. These preliminary studies have led to the finding that, as in adults, enzyme values are elevated in meningitis but that normal CSF lactic dehydrogenase levels are somewhat lower in children than in adults.

MATERIALS AND METHODS

The CSF of 81 children was obtained by lumbar puncture at Children's Hospital, George Washington University Hospital and the Walter Reed Army Medical Center. Selection of cases was exercised only in that CSF grossly xanthochromic or that obtained from traumatic lumbar puncture was rejected.

Conditions for assay of the lactic dehydrogenase (Reaction 1)

1) $CH_3COCOOH + DPNH + H^+ \rightleftharpoons CH_3CHOHCOOH + DPN^+$

content of CSF have been described in detail previously[4] and will only be summarized here. The reaction was conducted in a cuvette with 1.0 cm. light path in a volume of 1.0 ml. containing the following constituents: potassium phosphate at pH 7.5, 50 micromoles; DPNH, 0.1 micromole; CSF, 0.3 ml.; sodium pyruvate at pH 7.5, 5 micromoles. The reaction was initiated by the addition of pyruvate. Enzyme activity was followed at 340 mμ with a Beckman DU spectrophotometer at 30 second intervals for 3 minutes. A unit (μ) of lactic dehydrogenase activity is defined as that amount of lactic dehydrogenase which causes an optical density change of 0.001 per minute. Under these conditions enzyme activity is a linear function of time and of lactic dehydrogenase concentration.[4]

RESULTS

Patients in this study were evenly divided with respect to sex and included an age range of 1 day to 16 years. An approximately equal distribution was achieved among the following age groups: 0–2 years, 3–6 years and over 7 years. Patients have been grouped in five clinical categories with the results summarized in figure 1.

† The following abbreviations will be used: CSF, cerebrospinal fluid; DPN and DPNH, the oxidized and reduced forms of diphosphopyridine nucleotide, respectively; S.E., standard error of the mean.

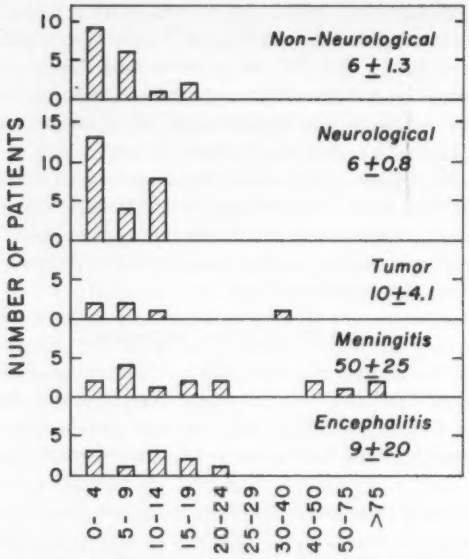

FIG. 1. Distribution of lactic dehydrogenase levels. The average value in μ and the standard error of the mean is presented for each series.

Nonneurological. Cases in this group include a variety of childhood diseases which were judged to have no neurological involvement. It must be cautioned, however, that since lumbar puncture is not a routine procedure on admission some suspicion of neurological involvement must have existed originally. One relatively high value (19μ) was that from the CSF of a malnourished infant who subsequently expired with pneumonia. The average level for 18 patients was 6μ.

Neurological. Patients with neurological disease other than meningitis, encephalitis and brain tumor were arbitrarily placed in this category. Included are cases with convulsions and mental retardation for which quantitative data is presented in table 1, as well as familial periodic paralysis (5μ), pseudotumor cerebri (3μ) and mongolism (8μ). An average of 6μ was found for 26 patients.

Tumor. Six cases of brain tumor included cerebellar astrocytoma (2μ, 5μ), brain stem tumor (8μ, 11μ, 28μ) and bilateral papilloma of the choroid plexus (3μ). An average of 10μ was obtained.

Encephalitis. Ten patients with a diagnosis of encephalitis had an average value of 10μ. These have been further subdivided according to etiology in

TABLE 1

Lactic Dehydrogenase in Several Disease States

Diagnosis	No. of Cases	Lactic Dehydrogenase (μ)	
		Mean	S.E.
Convulsions, afebrile	5	7	±2.0
Convulsions, febrile	3	2	±1.0
Mental retardation	9	5	±1.5
Encephalitis, measles	7	10	±2.9
Encephalitis, other	3	7	±2.8
Pneumonia (measles)	4	4	±1.5

table 1. For purposes of comparison cases of pneumonia complicating measles are included in table 1.

Meningitis. In a series of 16 patients with bacterial meningitis an average value of 50μ was found. It will be noted that lactic dehydrogenase values extended over a considerable range and included two very high (140μ, 410μ) titers.

DISCUSSION

Two points of interest become apparent from the results of this preliminary study. 1) When compared with adult titers the lactic dehydrogenase content of the CSF of children is slightly lower than that of adults. The mean values of 9.5μ and 6μ for adults and children, respectively, of neurological and nonneurological cases grouped together is a statistically significant difference, i.e., the possibility of the difference being ascribable to chance is less than one in twenty. 2) The lactic dehydrogenase titer of patients with meningitis is elevated. The values obtained in these cases are not, however, necessarily a function of the number of cells present in CSF nor of the protein concentration. Thus a value of 9μ was associated with a protein of 162 mg. per cent and 18,000 cells.

Because of the method of obtaining CSF it was not always possible to procure samples during the acute phase of the disease. For example, it is known that at least one of the samples (1μ) included under meningitis was taken three weeks after the acute episode and represents that of an essentially normal child. It is also necessary to comment on the high S.E. of the meningitis determinations which are due to two very high (140μ, 410μ) values. A comparison of the average titer obtained for meningitis in the present study with that of one conducted previously[4] indicated essentially identical findings, i.e., 50μ and 48μ.

It would appear that assay of CSF lactic dehydrogenase in acute cases

of suspected meningitis may add another parameter to its diagnosis and course. Lactic dehydrogenase was used as the indicator because of the ease of assay and freedom from errors. As has been pointed out previously[4] the apparently more popular indicator, transaminase, is subject to large errors owing to the presence of relatively high concentrations of pyruvate in CSF.

SUMMARY

The concentration of CSF lactic dehydrogenase has been determined in a series of children with a variety of disorders. Somewhat lower values were obtained with children than with adults. Elevated titers of lactic dehydrogenase were found in several acute cases of meningitis.

It is a pleasure to acknowledge the interest and cooperation of Dr. William B. Jakoby during the course of this work.

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Mushroom Poisoning

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Three children, all victims of accidental ingestion of mushrooms, were admitted to Children's Hospital during the month of August 1956. Two of the three were asymptomatic during a short hospital stay. In the third, whose case report follows, there developed mild symptoms consisting of nausea, vomiting, slight diarrhea and somnolence.

CASE REPORT

This was the first admission of a 13 month old white boy who, while playing in his back yard, ingested some wild mushrooms. Soon after swallowing them the patient started to perspire profusely, and vomited two to three times. Thirty minutes after

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ingestion, his stomach was lavaged of ingested material resembling mushrooms. Hospitalization was advised.

On admission physical examination, he was irritable and perspiring. The pupils were fixed and pin-point; pulse and respirations, rapid; and the bowel sounds, hyperactive. During the next 12 hours the patient continued to vomit, and passed two to three greenish black stools. Treatment consisted of clysis for dehydration and phenobarbital for restlessness. Twelve hours later his condition was remarkably improved and the patient was discharged.

DISCUSSION

Fungi form one of the largest groups of organisms in the plant kingdom. Included are the molds, mildews, rusts, mushrooms, and toadstools. Their effect on mankind can well be appreciated when their various uses are considered.

The vast armada of antibiotics derived from molds are only too familiar. Probably not so well appreciated is the important role fungi serve in the beer and wine industry.

Mushrooms have provided, for those so inclined, an eating delicacy for centuries. Records of cases of the ingestion of poisonous forms date back to ancient times. Early Roman writings relate the death of a pope and two emperors following the eating of poisonous species of mushrooms. Euripides' wife and three children died from similar causes.[1] Hippocrates, the father of medicine, relates the story of a young girl who showed severe gastrointestinal complaints following a meal of fungi. An emetic and a hot bath in this case were prescribed with excellent results.

Most of the information on mycetismus (mushroom poisoning) is contained in the literature of Europe where poisoning of this type is more common than in the United States. Our writings, however, although less in volume, supply more than enough material for a brief review of the subject.

In persons succumbing to the effects of mushroom poisoning, a characteristic history is usually encountered. The mushroom gatherer will usually rely on age-old, erroneous adages in distinguishing between poisonous and nonpoisonous varieties. A cap that peels or a plant that does not tarnish a silver coin on boiling have been given as proof that a mushroom is safely edible. Both these beliefs have no foundation in fact. *Amanita phalloides*, the most deadly mushroom known, will peel with ease.[2] The only way one can properly know whether or not a mushroom is safe for human consumption is by its proper anatomical identification. This usually requires the experience of an expert in the field of mycology.

Fungi can be divided into two large groups: 1) the family Agareceae or gill fungi (fig. 1); and 2) the family Polyporaceae or pore fungi. In the former the spores are produced on the plant's gills, whereas in the latter they are produced in minute tubes or pores. The poisonous varieties most often encountered are members of the gill fungi.

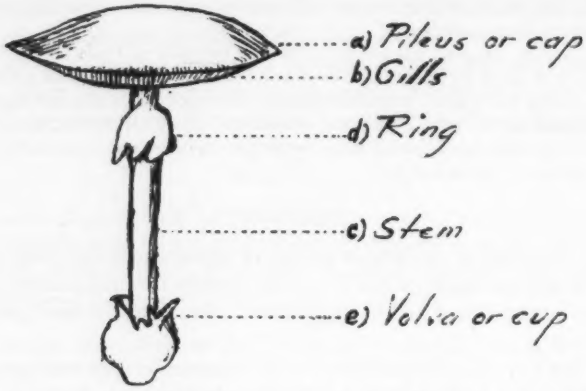

FIG. 1. A gill-bearing mushroom. Figure adapted from Circular 143, United States Department of Agriculture, March 1931.

The anatomy of the mushroom consists essentially of a cap or pileus, the gills or radiating plates on the undersurface of the pileus, and the stem. In some species there is a ring or annulus on the upper end of the stem and a volva or cuplike structure attached to the lower portion of the stem. The volva is sometimes called the "death cup" and serves as a distinguishing feature of the genus Amanita which contains the well known poisonous mushrooms.

At least 2,000 different varieties of mushrooms are present in this country alone; about 80 of these are considered poisonous. The varieties accounting for over 95 per cent of the morbidity and mortality are: a) *Amanita phalloides*—also called the death cap, and b) *A. muscaria*—also called the fly poison or fly agaric.

A. phalloides is well distributed throughout the United States and has accounted for over 90 per cent of the deaths resulting from mycetismus. The plant is usually seen growing in or along the border of woods, and frequently near the bases of oak trees. Its season is roughly from June to October. The mature plant is 4 to 5 inches in height and its cap 3½ inches in diameter. The color of the cap is white at first, and as the plant matures changes from a greenish brown to an olive gray. Both the gills and spores are white.

A. muscaria is second only in toxicity to *A. phalloides*. This variety is likewise widely distributed throughout this country, and occurs along the roadsides, wood margins and open woods, preferring a rather poor gravelly soil. Its season lasts from late spring to the first frost. It is described by botanists as a handsome plant 5 to 10 inches in height with a cap 4 to 6

inches in diameter. The cap is orange-red to yellow in color, and is characteristically covered with warts or scales. The gills are white to yellow and the spores white.

The symptomatology resulting from ingestion of these two plants differs and is directly related to the toxic principles contained in each.

The two main toxins isolated from *A. phalloides* are phalline and amanitine. The latter is the one responsible for symptoms and death. It is a heat-stable substance unaffected by cooking. Digestive juices likewise have no effect on its toxic nature, and it is easily absorbed.[1, 3]

The onset of clinically detectable symptoms consequent to eating the *A. phalloides* variety is characteristically slow. The symptoms generally do not appear until 6 to 24 hours after ingestion. After this initial quiescent period there is the sudden onset of colicky abdominal pain, nausea, vomiting and diarrhea. The stools frequently are bloody. During this phase, water and electrolyte losses are severe, and the danger of shock is grave.

Forty-eight hours after the onset of symptoms, signs of hepatic dysfunction begin to appear. The patient becomes increasingly jaundiced, and the liver is easily palpated and extremely tender to pressure. During this stage the patient may die consequent to acute liver failure. The pathology involved is a marked to an almost complete cellular destruction of the hepatic parenchyma.[4] The original architecture is almost completely replaced with fatty infiltrate. The amount of fat present has been estimated to be between 50 and 75 per cent of the total liver.[3]

Acute renal insufficiency, if it is to develop, occurs at about the seventy-second to the ninety-sixth hour. In patients dying during this stage the epithelium of the convoluted renal tubules is swollen and granular. In the same sections, double refractile fat bodies (cholesterol esters) have been demonstrated. Central nervous system abnormalities frequently develop, and convulsions and coma are not uncommon during the terminal stages of poisoning.

Treatment is supportive and is directed toward preventing shock and toward correcting fluid and electrolyte loss. The development of an effective antitoxin has apparently not been successful. In Europe, however, where this type of poisoning is more common, an antiphylloidin serum has been produced. According to some reports it has met with success provided the poisoning has been recognized early.[4] The French record good results in victims of this type poisoning by the oral administration of minced rabbit stomach.[4] The rabbit stomach, they claim, contains a neutralizing agent capable of detoxifying amanitine. Adrenal corticoids have been used but with inconclusive results.

A. muscaria is definitely not so toxic as the phalloides variety. The most familiar toxin contained in this particular plant, and the one on which

most experimentation has been done, is muscarine. Chemically it is a quaternary ammonium base closely related in structural configuration to choline. It is unaffected by heat or by any of the digestive juices.[1]

The onset of symptoms following ingestion of this species is rapid and will occur from within a few minutes to a few hours. Fortunately, in most cases vomiting occurs quickly and much of the plant is expelled. The rapidity of symptom development is undoubtedly a reason for this species' low mortality rate.

The symptoms encountered are directly related to stimulation of the autonomic nervous system. Nausea, vomiting and profuse diarrhea are swift in appearing. Excessive perspiration, lacrimation and salivation are present. The pupils are pin-point and do not react to light or accommodation. The pulse is slow and irregular. In more severe cases convulsions and coma develop. In fatal cases death occurs within 5 to 24 hours and is usually due to cardio-respiratory paralysis.

Fortunately an antidote is available and one should not hesitate to use it. Atropine sulfate administered intramuscularly or intravenously in what would normally be considered toxic doses proves effective in counteracting muscarinic symptoms. Supportive treatment in the way of fluid and electrolyte replacement is frequently necessary.

SUMMARY

Mushroom poisoning is of rare occurrence in this country. The ingestion of poisonous species, however, does occur.

The two varieties accounting for over 95 per cent of the mortality and morbidity resulting from mycetismus belong to the genus Amanita. Both these species can be found growing in almost any section of this country.

A. phalloides, the more deadly of the two, accounts for over 90 per cent of the deaths resulting from mycetismus. The onset of symptoms is slow, occurring 6 to 24 hours after ingestion. They consist of nausea, vomiting and profuse diarrhea followed by hepatorenal failure with or without accompanying central nervous system dysfunction. Therapy is mainly supportive.

A. muscaria does not generally cause death. Symptoms are related to overstimulation of the autonomic nervous system and are rapid in development. Treatment is supportive plus the use of atropine, the specific antidote for this type of poisoning.

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Clinical Brief: a Case of Cat Scratch Disease Complicated by Erythema Nodosum

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This case is presented to emphasize the fact that cat scratch disease may present with secondary manifestations. The progressive nature of the skin lesions in this case was primarily responsible for hospitalization for diagnostic work-up.

Of six cases of cat scratch disease in which a positive diagnosis has been made at Children's Hospital,[1] this is the only one in which erythema nodosum was a complication, and the only case in which any skin manifestation was noted.

CASE REPORT

H. P., a 4 year old white girl, was admitted to Children's Hospital November 29, 1957, because of a mass in the right axilla and skin lesions on both lower extremities. Ten days prior to admission, a soft lump the size of a walnut in the patient's axilla was first noted by the child's mother. No erythema, however, was noted. Seven days prior to admission, the patient complained of pain in the area of the mass and was therefore taken to see her family physician. At that time her temperature was 100° F. The physician noted the axillary mass described and an inflamed pharynx. The patient was given an injection of penicillin and sulfonamide by mouth. The following day, tender reddish nodules which did not itch were noted on both lower extremities below the knees. The sulfonamide was stopped and the patient was started on Mysteclin®. The child had a persistently poor appetite until admission. Her temperature remained elevated to 100°F. No other constitutional symptoms were noted. In spite of therapy, the axillary mass continued to increase in size and the skin lesions increased in number. For this reason, hospitalization for diagnostic work-up was advised.

Investigation of the environment revealed that the patient lived on a farm. She drank

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FIG. 1. Erythema nodosum

well water and raw milk daily. According to the mother, the milk came from one cow which was tested regularly according to state regulations. This child had also continuous contact with domesticated rabbits and chickens. There were two cats around the farm and the patient had played with them frequently. However, no specific history of a scratch had been obtained.

Admission physical examination revealed a well developed, well nourished alert 4 year old white girl who did not appear acutely ill. Her temperature was 99°F., pulse rate was 80 and respiratory rate was 20. There were two small healing scratch marks on the abdomen and a small healing scratch mark on the right wrist. Scattered over the lower extremities to just above the knees, there were multiple, slightly raised, round, erythematous nodules varying in size from 1 to 5 cm. in diameter (fig. 1). The edges were not sharply demarcated. A slight increase in temperature was noted in the lesions. There was a firm, tender, smooth, 2 by 3 cm. mass in the right axilla (fig. 2). A small fluctuant area was noted in the center of the mass. There was no increase in heat or erythema. An epitrochlear gland on the right was palpable and measured 1 by 1 cm.

The blood count on admission revealed a hemoglobin of 9.9 Gm. per 100 ml. and a white count of 15,400 with 74 per cent segmented forms, 1 per cent eosinophiles, 1 per cent basophiles, 23 per cent lymphocytes and 1 per cent monocytes. The hematocrit was 35 and platelets were adequate. Urine examination was normal. The corrected

FIG. 2. Enlarged right axillary lymph node. Note positive reaction to cat scratch antigen on left forearm.

sedimentation rate was 41 mm. per hour. VDRL was negative. Agglutination tests gave the following results: typhoid H negative, salmonella groups A and B negative, salmonella group C 1:20, salmonella group D negative, salmonella group E 1:80, paratyphoid groups A, B and C negative, proteus OX-19 negative and *B. tularensis* negative. Heterophile agglutination was positive 1:28. Total protein was 8 Gm. per 100 ml., with an albumin of 3.5 Gm. per 100 ml. and globulin 4.5 Gm. per 100 ml.

On admission, PPD, intermediate strength, and histoplasmin tests were applied. These were both negative. A chest x-ray showed no evidence of pulmonary disease. On November 30, the child was seen by a consultant who felt that cat scratch disease should be strongly considered. Twenty-four hours after intradermal injection of cat scratch antigen, there was an area of erythema 2.0 cm. in diameter with a central papule measuring 0.5 cm. present on the left forearm (see figure 2). This was regarded as a positive reaction. On this day, a central area of erythema was noted over the axillary mass and the leg lesions appeared to be fading.

The patient remained afebrile throughout her hospital stay although no antibiotics were given. It was felt that she was improving and she was therefore discharged to be followed by her private physician.

COMMENT

Of 160 cases of cat scratch disease reviewed by Daniels and MacMurray,[2] all had positive skin tests to cat scratch antigen; skin manifestations were noted in 11 patients. The rash was maculopapular in 6, erythematous in 1

and finely papular in 2. The remaining 2 were complicated by erythema nodosum. In one of these, the lesions were characteristic and lasted three weeks.

It is hoped that cat scratch disease will be kept in mind in any differential diagnosis of lymph node lesions and especially in those cases in which erythema nodosum is a manifestation.

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The Editor's Column

OPERATING TIME IS IMPORTANT

The wide range of safety present in adult surgery is not to be found in pediatric surgery. In the child the pre-anesthetic pulse rate of 140–160 may increase to 200–250 under anesthesia and may be too fast to be palpably measured. Such an increase under anesthesia may be seen in children up to the age of 10 years. These fast pulses cannot be sustained for long periods. The technical difficulty of maintaining adequate pulmonary ventilation is always great under the age of 3 years because of the reduced tidal air exchange and labile respiratory rate. The loss of 30 cc. of blood may be equivalent to the loss of 500 cc. in the adult. Such direct bleeding or loss of fluid into traumatized exposed tissues may seriously reduce the circulating blood volume.

Undetected anemia may be quite a handicap to the maintenance of proper oxygenation of the tissues. Circulating anti-bodies which may be very high at birth soon decrease, and the circulating gamma globulins may not rise to adequate levels until after 3 to 6 months of age. Resistance to infection at this time is normally definitely lowered and is further reduced by the prolonged exposure of tissues occurring during surgical procedures.

All the foregoing make the operating time in the surgery of the infant and young child quite important. Safe deliberate speed in executing a surgical procedure is a prime factor in keeping down the morbidity and mortality in pediatric surgery. Multiple-stage procedures rather than prolonged single-stage procedures should be considered.

J. H. B.

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